```
=> Biotinylation
L1          3322 BIOTINYLATION

=> "viral  antigen"
L2          9232 "VIRAL  ANTIGEN"

=> L1 and L2
L3             3 L1 AND L2

=> "viral envelope protein"
L4          1012 "VIRAL ENVELOPE PROTEIN"

=> L1 and L4
L5             1 L1 AND L4

=> "viral surface protein"
L6           138 "VIRAL SURFACE PROTEIN"

=> L1 and L6
L7             0 L1 AND L6

=> influenza and L1
L8            19 INFLUENZA AND L1

=> HIV and L1
L9            54 HIV AND L1

=> gp120 and L9
L10            7 GP120 AND L9

=> gp160 and L1
L11           10 GP160 AND L1

=> D L3 IBIB TI AU ABS 1-
```

DOCUMENT NUMBER: PREV199497549929
TITLE: Complete inactivation of target mRNA by biotinylated antisense oligodeoxynucleotide-avidin conjugates.
AUTHOR(S): Boado, Ruben J. [Reprint author]; Pardridge, William M.
CORPORATE SOURCE: Dep. Med., Brain Res. Inst., UCLA Sch. Med., Los Angeles, CA 90024, USA
SOURCE: Bioconjugate Chemistry, (1994) Vol. 5, No. 5, pp. 406-410. CODEN: BCCHES. ISSN: 1043-1802.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Dec 1994
Last Updated on STN: 15 Dec 1994

TI Complete inactivation of target mRNA by biotinylated antisense oligodeoxynucleotide-avidin conjugates.

SO Bioconjugate Chemistry, (1994) Vol. 5, No. 5, pp. 406-410. CODEN: BCCHES. ISSN: 1043-1802.

AU Boado, Ruben J. [Reprint author]; Pardridge, William M.

AB **Biotinylation** of phosphodiester oligodeoxynucleotides (PO-ODN) allows for conjugation to avidin-based transcellular delivery systems. In addition, **biotinylation** of PO-ODN at the 3'-terminus provides complete protection against serum 3'-exonuclease degradation. The present study was undertaken to determine if antisense 3'-biotinylated PO-ODN-avidin constructs are able to recognize and inactivate the target mRNA through RNase H-mediated degradation. A 21-mer antisense PO-ODN complementary to the tat gene encompassing nucleotides 5402-5422 of the **HIV**-1 genome was synthesized with biotin conjugated to the 3'-terminus (bio-tat). Gel mobility assays using (5'-32P)-labeled bio-tat ODN and avidin showed that the bio-tat ODN was fully monobiotinylated. Aliquots of (32P)-labeled sense or antisense tat RNA (337 and 351 nucleotides, respectively) were prepared from transcription plasmids and were preincubated with an excess of bio-tat ODN with or without avidin constructs and digested with RNase H. Products were resolved with sequencing gel and analyzed by autoradiography. Complete conversion to predicted RNA fragments resulting from RNase H digestion of the RNA-ODN duplex (53 and 263 nucleotides) was observed when (32P)-tat sense RNA was incubated with antisense bio-tat ODN or conjugated to avidin or an avidin-cationized human serum albumin (cHSA) complex. Conversely, no degradation of (32P)-tat-antisense RNA was observed after incubation with antisense bio-tat ODN and RNase H. In addition, the avidin-cHSA complex significantly increased (84-fold) the uptake of (32P)-internally labeled bio-tat ODN and its stability against cellular nuclease degradation in peripheral blood lymphocytes. In conclusion, biotinylated antisense ODN-avidin constructs induce complete inactivation of target mRNA by RNase H. Therefore, 3'-biotinylated PO-ODNs have the advantages of (a) resistance to serum and cellular 3'-exonuclease, (b) conjugation by avidin-based transcellular delivery systems, and (c) inactivation of target mRNA via RNase H degradation.

L9 ANSWER 48 OF 54 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:406124 BIOSIS
DOCUMENT NUMBER: PREV199800406124
TITLE: Retention of the human immunodeficiency virus type 1 envelope glycoprotein in the endoplasmic reticulum does not redirect virus assembly from the plasma membrane.
AUTHOR(S): Salzwedel, Karl; West, John T., Jr.; Mulligan, Mark J.; Hunter, Eric [Reprint author]
CORPORATE SOURCE: Dep. Microbiol., Univ. Alabama at Birmingham, 845 19th St. South, Birmingham, AL 35294-2170, USA
SOURCE: Journal of Virology, (Sept., 1998) Vol. 72, No. 9, pp. 7523-7531. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Sep 1998
Last Updated on STN: 21 Sep 1998

TI Retention of the human immunodeficiency virus type 1 envelope glycoprotein in the endoplasmic reticulum does not redirect virus assembly from the plasma membrane.

SO Journal of Virology, (Sept., 1998) Vol. 72, No. 9, pp. 7523-7531. print.
CODEN: JOVIAM. ISSN: 0022-538X.

AU Salzwedel, Karl; West, John T., Jr.; Mulligan, Mark J.; Hunter, Eric [Reprint author]

AB The envelope glycoprotein (Env) of human immunodeficiency virus type 1 (**HIV**-1) has been shown to redirect the site of virus assembly in polarized epithelial cells. To test whether localization of the glycoprotein exclusively to the endoplasmic reticulum (ER) could redirect virus assembly to that organelle in nonpolarized cells, an ER -retrieval signal was engineered into an epitope-tagged variant of Env. The epitope tag, attached to the C terminus of Env, did not affect the normal maturation and transport of the glycoprotein or the incorporation of Env into virions. The epitope-tagged Env was also capable of mediating syncytium formation and virus entry with a similar efficiency to that of wild-type Env. When the epitope was modified to contain a consensus K(X)KXX ER retrieval signal, however, the glycoprotein was no longer proteolytically processed into its surface and transmembrane subunits and Env could not be detected at the cell surface by **biotinylation**. Endoglycosidase H analysis revealed that the modified Env was not transported to the Golgi apparatus. Immunofluorescent staining patterns were also consistent with the exclusion of Env from the Golgi. As expected, cells expressing the modified Env failed to form syncytia with CD4+ permissive cells. Despite this tight localization of Env to the ER, when the modified Env was expressed in the context of virus, virions continued to be produced efficiently from the plasma membrane of transfected cells. However, these virions contained no detectable glycoprotein and were noninfectious. Electron microscopy revealed virus budding from the plasma membrane of these cells, but no virus was seen assembling at the ER membrane and no assembled virions were found within the cell. These results suggest that the accumulation of Env in an intracellular compartment is not sufficient to redirect the assembly of **HIV** Gag in nonpolarized cells.

L9 ANSWER 30 OF 54 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:506400 CAPLUS
DOCUMENT NUMBER: 121:106400
TITLE: Human immunodeficiency virus type 1 Nef-induced down-modulation of CD4 is due to rapid internalization and degradation of surface CD4
AUTHOR(S): Rhee, Sung S.; Marsh, Jon W.
CORPORATE SOURCE: Laboratory of Molecular Biology, Natl. Institute of Mental Health, Bethesda, MD, 20892, USA
SOURCE: Journal of Virology (1994), 68(8), 5156-63
CODEN: JOVIAM; ISSN: 0022-538X
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Human immunodeficiency virus type 1 Nef-induced down-modulation of CD4 is due to rapid internalization and degradation of surface CD4

SO Journal of Virology (1994), 68(8), 5156-63
CODEN: JOVIAM; ISSN: 0022-538X

AU Rhee, Sung S.; Marsh, Jon W.

AB Human immunodeficiency virus type 1 (**HIV**-1) Nef is a myristoylated protein with a relative mol. mass of 27 kDa, is localized to the cytoplasmic surfaces of cellular membranes, and has been reported to down-modulate CD4 in human T cells. To understand the mechanism of **HIV**-1 Nef-mediated down-modulation of cell surface CD4, the authors expressed Nef protein in human T-cell line VB. Expression of **HIV**-1 Nef protein down-modulated surface CD4 mols. In pulse-chase expts., CD4 mols. in Nef-expressing cells were synthesized at normal levels. However, the bulk of newly synthesized CD4 protein was degraded with a half-life of approx. 6 h, compared with the 24-h half-life in control cells. This Nef-induced acceleration of CD4 turnover was inhibited by lysosomotropic agents NH4Cl and chloroquine as well as by the protease inhibitor leupeptin. Surface CD4 **biotinylation** expts. demonstrated that CD4 mols. in Nef-expressing T cells are transported to the plasma membrane with normal kinetics but are then rapidly internalized. Therefore, **HIV**-1 Nef-induced down-modulation of CD4 is due to rapid internalization of surface CD4 and subsequent degradation by an acid-dependent process, potentially lysosomal. Addnl., in a Nef-expressing cell, the authors find accelerated dissociation of the T-cell tyrosine kinase p56lck and CD4 but only after the complex reaches the plasma membrane. This implies that **HIV**-1 Nef protein might play a role in triggering a series of T-cell activation-like events, which contribute to p56lck dissociation and internalization of surface CD4 mols.

L9 ANSWER 28 OF 54 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:628630 CAPLUS
DOCUMENT NUMBER: 121:228630
TITLE: Altered glycosylation of leukosialin, CD43, in **HIV**-1-infected cells of the CEM line
AUTHOR(S): Lefebvre, Jean-Claude; Giordanengo, Valerie; Limouse, Martine; Doglio, Alain; Cucchiarini, Magali; Monpoux, Fabrice; Mariani, Roger; Peyron, Jean-Francois
CORPORATE SOURCE: Lab. Virol., Unite Inst. Natl. Sante Rech. Med. (INSERM), Nice, 06107, Fr.
SOURCE: Journal of Experimental Medicine (1994), 180(5), 1609-17
CODEN: JEMEAV; ISSN: 0022-1007
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Altered glycosylation of leukosialin, CD43, in **HIV**-1-infected cells of the CEM line
SO Journal of Experimental Medicine (1994), 180(5), 1609-17
CODEN: JEMEAV; ISSN: 0022-1007
AU Lefebvre, Jean-Claude; Giordanengo, Valerie; Limouse, Martine; Doglio, Alain; Cucchiarini, Magali; Monpoux, Fabrice; Mariani, Roger; Peyron, Jean-Francois
AB CD43 (leukosialin, gpL115, sialophorin) is a major sialoglycoprotein widely expressed on hematopoietic cells that is defective in the congenital immunodeficiency Wiskott-Aldrich syndrome. It is thought to play an important role in cell-cell interactions and to be a costimulatory mol. for T lymphocyte activation. Using a metabolic 35SO42- radiolabeling assay or **biotinylation** of cell surface proteins, the authors discovered that CD43 are sulfated mols. the glycosylation of which is altered in human immunodeficiency virus type 1 (**HIV**-1)-infected leukemic T cells of the CEM line. Hyposialylation of O-glycans and changed substitution on N-acetylgalactosamine residues are observed The glycosylation defect is associated with an impairment of CD43-mediated homotypic aggregation which can be restored by resialylation. The hyposialylation of CD43 on **HIV**-1+ cells may explain the high prevalence of autoantibodies directed against nonsialylated CD43 that have been detected in **HIV**-1-infected individuals. A defect in glycosylation of important mols. such as CD43 or, as recently described, CD45 may explain alterations of T cell functions and viability in **HIV**-1-infected individuals. In addition, a possible implication of hyposialylation in the **HIV**-1-infected cells entrapment in lymph nodes could be envisioned.

ACCESSION NUMBER: 1998:540537 CAPLUS
DOCUMENT NUMBER: 129:257507
TITLE: Retention of the human immunodeficiency virus type 1 envelope glycoprotein in the endoplasmic reticulum does not redirect virus assembly from the plasma membrane
AUTHOR(S): Salzwedel, Karl; West, John T., Jr.; Mulligan, Mark J.; Hunter, Eric
CORPORATE SOURCE: Department of Microbiology, University of Alabama at Birmingham, Birmingham, AL, 35294, USA
SOURCE: Journal of Virology (1998), 72(9), 7523-7531
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Retention of the human immunodeficiency virus type 1 envelope glycoprotein in the endoplasmic reticulum does not redirect virus assembly from the plasma membrane

SO Journal of Virology (1998), 72(9), 7523-7531
CODEN: JOVIAM; ISSN: 0022-538X

AU Salzwedel, Karl; West, John T., Jr.; Mulligan, Mark J.; Hunter, Eric

AB The envelope glycoprotein (Env) of human immunodeficiency virus type 1 (**HIV**-1) has been shown to redirect the site of virus assembly in polarized epithelial cells. To test whether localization of the glycoprotein exclusively to the endoplasmic reticulum (ER) could redirect virus assembly to that organelle in nonpolarized cells, an ER-retrieval signal was engineered into an epitope-tagged variant of Env. The epitope tag, attached to the C terminus of Env, did not affect the normal maturation and transport of the glycoprotein or the incorporation of Env into virions. The epitope-tagged Env was also capable of mediating syncytium formation and virus entry with a similar efficiency to that of wild-type Env. When the epitope was modified to contain a consensus K(X)KXX ER retrieval signal, however, the glycoprotein was no longer proteolytically processed into its surface and transmembrane subunits and Env could not be detected at the cell surface by **biotinylation**. Endoglycosidase H anal. revealed that the modified Env was not transported to the Golgi apparatus Immunofluorescent staining patterns were also consistent with the exclusion of Env from the Golgi. As expected, cells expressing the modified Env failed to form syncytia with CD4+ permissive cells. Despite this tight localization of Env to the ER, when the modified Env was expressed in the context of virus, virions continued to be produced efficiently from the plasma membrane of transfected cells. However, these virions contained no detectable glycoprotein and were noninfectious. Electron microscopy revealed virus budding from the plasma membrane of these cells, but no virus was seen assembling at the ER membrane and no assembled virions were found within the cell. These results suggest that the accumulation of Env in an intracellular

ACCESSION NUMBER: 1999:167527 CAPLUS
DOCUMENT NUMBER: 131:70988
TITLE: Processing and routage of **HIV** glycoproteins by furin to the cell surface
AUTHOR(S): Moulard, Maxime; Hallenberger, Sabine; Garten, Wolfang; Klenk, Hans-Dieter
CORPORATE SOURCE: Centre d'Immunologie de Marseille Luminy, Marseille, Fr.
SOURCE: Virus Research (1999), 60(1), 55-65
CODEN: VIREDF; ISSN: 0168-1702
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Processing and routage of **HIV** glycoproteins by furin to the cell surface

SO Virus Research (1999), 60(1), 55-65
CODEN: VIREDF; ISSN: 0168-1702

AU Moulard, Maxime; Hallenberger, Sabine; Garten, Wolfang; Klenk, Hans-Dieter

AB Proteolytic activation of **HIV**-1 and **HIV**-2 envelope glycoprotein precursors (gp160 and gp140, resp.) occurs at the carboxyl side of a consensus motif consisting of the highly basic amino acid sequence. We have shown previously () and confirmed in this report, that furin and PC7 can be considered as the putative physiol. enzymes involved in the proteolytic activation of the **HIV**-1 and **HIV**-2 envelope precursors. In this study, we show by cell surface **biotinylation** and immunopptn. of the cell surface associated viral glycoproteins with antibodies that the mature viral envelope glycoproteins are correctly transported to the cell membrane. Furthermore, we show that the uncleaved forms of the glycoproteins (gp160HIV-1 and gp140HIV-2) are also highly represented at the cell surface. First, transient expression of gp160 and gp140 into CV1, a cell line known to be inefficient in the proteolytic processing of the env gene, results in the expression of gp160 and gp140 at the cell surface. Moreover, **HIV**-1 infection of T cells also showed that gp160 is directed to the cell surface. In addition, the authors show that the precursor is not incorporated in the virus particle following the budding from the cell surface. Furthermore, a gp160 mutant (deficient for three carbohydrate sites on the gp41), shown to be poorly processed with the coexpressed endoproteases, is found to be transported as an uncleaved precursor to the cell surface. In contrast to **HIV** envelope glycoproteins, the influenza hemagglutinin precursor (HA0), that is thought to be matured by the furin-like enzymes as well, is found to be retained within the cell and is not able to reach the cell surface. These results show that the proteolytic maturation of the viral envelope precursors of human immunodeficiency viruses type 1 and type 2 is not a prerequisite for cell surface targeting of the **HIV** glycoproteins. Implications of these results for antiviral immune response are discussed.

L8 ANSWER 15 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:47203 BIOSIS
DOCUMENT NUMBER: PREV199900047203
TITLE: Use of the ARPE-19 cell line as a model of RPE polarity: Basolateral secretion of FGF5.
AUTHOR(S): Dunn, Kerrin C.; Marmorstein, Alan D.; Bonilba, Vera L.; Rodriguez-Boulan, Enrique; Giordano, Frank; Hjelmeland, Leonard M. [Reprint author]
CORPORATE SOURCE: Vitreoretinal Res. Lab., Sch. Med., Univ. Calif., One Shields Ave., Davis, CA 95616-8794, USA
SOURCE: IOVS, (Dec., 1998) Vol. 39, No. 13, pp. 2744-2749. print.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Feb 1999
Last Updated on STN: 10 Feb 1999

TI Use of the ARPE-19 cell line as a model of RPE polarity: Basolateral secretion of FGF5.

SO IOVS, (Dec., 1998) Vol. 39, No. 13, pp. 2744-2749. print.

AU Dunn, Kerrin C.; Marmorstein, Alan D.; Bonilba, Vera L.; Rodriguez-Boulan, Enrique; Giordano, Frank; Hjelmeland, Leonard M. [Reprint author]

AB PURPOSE. To determine the polarity of fibroblast growth factor 5 (FGF5) secretions from retinal pigment epithelium (RPE) cells and to examine the viability and utility of the ARPE-19 cell line as a model for the study of RPE polarity. METHODS. **Influenza** infection and adenovirus-mediated gene transfer were used to deliver and express genes encoding **influenza** hemagglutinin (HA), p75-NTR (a neurotrophin receptor), low-density lipoprotein (LDL) receptor (LDLR), and FGF5 in confluent monolayers of ARPE-19 cells. The localization of HA, p75-NTR, and LDLR was determined by confocal microscopy. Domain selective **biotinylation** assays were used to quantitatively determine the polarities of p75-NTR and LDLR. The secretion of FGF5 into the apical and basal media of ARPE-19 cultures was examined by immunoblot analysis of conditioned media. RESULTS. Hemagglutinin and p75-NTR were found to be localized on the apical surface of infected and transduced ARPE-19 cells. In contrast, LDLR was associated preferentially with the basolateral membrane of ARPE-19 cells. **Biotinylation** studies indicated that 84% of p75-NTR was present on the apical surface, and 79% of LDLR was basolaterally polarized. Over the course of 6 hours, more than 90% of the total secreted FGF5 protein accumulated in the basolateral media. CONCLUSIONS. ARPE-19 cells exhibit a polarized distribution of cell surface markers when examined by either confocal microscopy or surface-labeling assays. This indicates that the ARPE-19 cell line is a valid model for studies of RPE cell polarity. FGF5, a secreted protein normally produced by RPE cells, is accumulated preferentially in the basal media after only 6 hours, suggesting that it is vectorially secreted from the basolateral surface of ARPE-19 cells.

L8 ANSWER 13 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:391802 BIOSIS
DOCUMENT NUMBER: PREV199900391802
TITLE: Polarity of osteoblasts and osteoblast-like UMR-108 cells.
AUTHOR(S): Ilvesaro, Joanna [Reprint author]; Metsikko, Kalervo; Vaananen, Kalervo; Tuukkanen, Juha
CORPORATE SOURCE: Department of Anatomy, University of Oulu, Kajaanintie 52A, FIN-90220, Oulu, Finland
SOURCE: Journal of Bone and Mineral Research, (Aug., 1999) Vol. 14, No. 8, pp. 1338-1344. print.
CODEN: JBMREJ. ISSN: 0884-0431.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Sep 1999
Last Updated on STN: 28 Sep 1999

TI Polarity of osteoblasts and osteoblast-like UMR-108 cells.

SO Journal of Bone and Mineral Research, (Aug., 1999) Vol. 14, No. 8, pp. 1338-1344. print.
CODEN: JBMREJ. ISSN: 0884-0431.

AU Ilvesaro, Joanna [Reprint author]; Metsikko, Kalervo; Vaananen, Kalervo; Tuukkanen, Juha

AB Enveloped viruses, such as vesicular stomatitis virus (VSV) and **Influenza** virus, have been widely used in studying epithelial cell polarity. Viral particles of VSV-infected epithelial cells bud from the basolateral membrane, which is in contact with the internal milieu and the blood supply. **Influenza**-infected cells bud viral particles from the apical surface facing the external milieu. This feature can be utilized in labeling polarized membrane domains. We studied the polarity of mesenchymal osteoblasts using osteosarcoma cell line UMR-108 and endosteal osteoblasts in situ in bone tissue cultures. Immunofluorescence confocal microscopy revealed that the VSV glycoprotein (VSV G) was targeted to the culture medium-facing surface. In endosteal osteoblasts, VSV G protein was found in the surface facing bone marrow and circulation. On the contrary, **Influenza** virus hemagglutinin (HA) was localized to the bone substrate-facing surface of the UMR-108 cells. Electron microscopy showed that in the cases where the cells were growing as a single layer, VSV particles were budding from the culture medium-facing surface, whereas **Influenza** viruses budded from the bone substrate-facing surface. When the cells overlapped, this polarity was lost. Cell surface **biotinylation** revealed that 55% of VSV G protein was biotinylated, whereas **Influenza** virus HA was only 22% biotinylated. These findings suggest that osteoblasts are polarized at some point of their life cycle. The bone-attaching plasma membrane of osteoblasts is apical, and the circulation or bone marrow-facing plasma membrane is basolateral in nature.

L8 ANSWER 12 OF 19 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:122577 BIOSIS
DOCUMENT NUMBER: PREV200000122577
TITLE: MAL, an integral element of the apical sorting machinery, is an itinerant protein that cycles between the trans-Golgi network and the plasma membrane.
AUTHOR(S): Puertollano, Rosa; Alonso, Miguel A. [Reprint author]
CORPORATE SOURCE: Centro de Biologia Molecular "Severo Ochoa," Consejo Superior de Investigaciones Cientificas, Universidad Autonoma de Madrid, Cantoblanco, 28049, Madrid, Spain
SOURCE: Molecular Biology of the Cell, (Oct., 1999) Vol. 10, No. 10, pp. 3435-3447. print.
CODEN: MBCEEV. ISSN: 1059-1524.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Apr 2000
Last Updated on STN: 3 Jan 2002

TI MAL, an integral element of the apical sorting machinery, is an itinerant protein that cycles between the trans-Golgi network and the plasma membrane.

SO Molecular Biology of the Cell, (Oct., 1999) Vol. 10, No. 10, pp. 3435-3447. print.
CODEN: MBCEEV. ISSN: 1059-1524.

AU Puertollano, Rosa; Alonso, Miguel A. [Reprint author]

AB The MAL proteolipid is a nonglycosylated integral membrane protein found in glycolipid-enriched membrane microdomains. In polarized epithelial Madin-Darby canine kidney cells, MAL is necessary for normal apical transport and accurate sorting of the **influenza** virus hemaglutinin. MAL is thus part of the integral machinery for glycolipid-enriched membrane-mediated apical transport. At steady state, MAL is predominantly located in perinuclear vesicles that probably arise from the trans-Golgi network (TGN). To act on membrane traffic and to prevent their accumulation in the target compartment, integral membrane elements of the protein-sorting machinery should be itinerant proteins that cycle between the donor and target compartments. To establish whether MAL is an itinerant protein, we engineered the last extracellular loop of MAL by insertion of sequences containing the FLAG epitope or with sequences containing residues that became O-glycosylated within the cells or that displayed biotinylatable groups. The ectopic expression of these modified MAL proteins allowed us to investigate the surface expression of MAL and its movement through different compartments after internalization with the use of a combination of assays, including surface **biotinylation**, surface binding of anti-FLAG antibodies, neuraminidase sensitivity, and drug treatments. Immunofluorescence and flow cytometric analyses indicated that, in addition to its Golgi localization, MAL was also expressed on the cell surface, from which it was rapidly internalized. This retrieval implies transport through the endosomal pathway and requires endosomal acidification, because it can be inhibited by drugs such as chloroquine, monensin, and NH4Cl. Resialylation experiments of surface MAL treated with neuraminidase indicated that apprx30% of the internalized MAL molecules were delivered to the TGN, probably to start a new cycle of cargo transport. Together, these observations suggest that, as predicted for integral membrane members of the late protein transport machinery, MAL is an itinerant protein cycling between the TGN and the plasma membrane.

L8 ANSWER 10 OF 19 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:211876 CAPLUS
DOCUMENT NUMBER: 116:211876
TITLE: Opposite polarity of virus budding and of viral envelope glycoprotein distribution in epithelial cells derived from different tissues
AUTHOR(S): Zurzolo, Chiara; Polistina, Claudio; Saini, Marco; Gentile, Raffaele; Aloj, Luigi; Migliaccio, Giovanni; Bonatti, Stefano; Nitsch, Lucio
CORPORATE SOURCE: Cent. Endocrinol. Oncol. Sper., Cons. Naz. Ric., Naples, 80131, Italy
SOURCE: Journal of Cell Biology (1992), 117(3), 551-64
CODEN: JCLBA3; ISSN: 0021-9525
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Opposite polarity of virus budding and of viral envelope glycoprotein distribution in epithelial cells derived from different tissues

SO Journal of Cell Biology (1992), 117(3), 551-64
CODEN: JCLBA3; ISSN: 0021-9525

AU Zurzolo, Chiara; Polistina, Claudio; Saini, Marco; Gentile, Raffaele; Aloj, Luigi; Migliaccio, Giovanni; Bonatti, Stefano; Nitsch, Lucio

AB The surface envelope glycoprotein distribution and the budding polarity of four RNA viruses in Fischer rat thyroid (FRT) cells and in CaCo-2 cells derived from a human colon carcinoma were compared. Whereas both FRT and CaCo-2 cells sort similarly **influenza** hemagglutinin and vesicular stomatitis virus (VSV) G protein, resp., to apical and basolateral membrane domains, they differ in their handling of two togaviruses Sinbis and Semiliki Forest virus (SFV). By conventional FM, Sindbis virus and SFV bud apically in FRT cells and basolaterally in CaCo-2 cells. Consistent with this finding, the distribution of the p62/E2 envelope glycoprotein of SFV, assayed by immuno-electron microscopy and by domain-selective surface **biotinylation** was predominantly apical on FRT cells and basolateral on CaCo-2 cells. A given virus and its envelope glycoprotein can be delivered to opposite membrane domains in epithelial cells derived from different tissues. The tissue specificity in the polarity of virus budding and viral envelope glycoprotein distribution indicate that the sorting machinery varies considerably between different epithelial cell types.

L10 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1994:161617 CAPLUS
DOCUMENT NUMBER: 120:161617
TITLE: Process for the determination of peptides corresponding to immunologically important epitopes and their use in a process for determination of antibodies, or biotinylated peptides corresponding to immunologically important epitopes, a process for preparing them and compositions containing them
INVENTOR(S): De Leys, Robert
PATENT ASSIGNEE(S): N.V. Innogenetics S.A., Belg.
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9318054 | A2 | 19930916 | WO 1993-EP517 | 19930308 |
| WO 9318054 | A3 | 19940217 | | |
| W: AU, BB, BG, BR, CA, CZ, FI, HU, JP, KP, KR, LK, MG, MN, MW, NO, NZ, PL, PT, RO, RU, SD, SK, UA, US | | | | |
| RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, SN, TD, TG | | | | |
| EP 564746 | A1 | 19931013 | EP 1992-400598 | 19920306 |
| R: BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE | | | | |
| CA 2102301 | AA | 19930907 | CA 1993-2102301 | 19930308 |
| AU 9337463 | A1 | 19931005 | AU 1993-37463 | 19930308 |
| AU 671623 | B2 | 19960905 | | |
| EP 589004 | A1 | 19940330 | EP 1993-906490 | 19930308 |
| EP 589004 | B1 | 19990506 | | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE | | | | |
| JP 06505806 | T2 | 19940630 | JP 1993-515334 | 19930308 |
| JP 3443809 | B2 | 20030908 | | |
| BR 9305435 | A | 19941227 | BR 1993-5435 | 19930308 |
| EP 891982 | A2 | 19990120 | EP 1998-202777 | 19930308 |
| EP 891982 | A3 | 20000412 | | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE | | | | |
| AT 179716 | E | 19990515 | AT 1993-906490 | 19930308 |
| ES 2133392 | T3 | 19990916 | ES 1993-906490 | 19930308 |
| US 5891640 | A | 19990406 | US 1993-146028 | 19931122 |
| US 6165730 | A | 20001226 | US 1996-723425 | 19960930 |
| US 6210903 | B1 | 20010403 | US 1998-112206 | 19980709 |
| US 6667387 | B1 | 20031223 | US 2000-576824 | 20000523 |
| US 6649735 | B1 | 20031118 | US 2001-790497 | 20010223 |
| JP 2004002379 | A2 | 20040108 | JP 2003-107716 | 20030411 |

PRIORITY APPLN. INFO.:

| | | |
|---|---|---|
| EP 1992-400598 | A | 19920306 |
| EP 1993-906490 | A3 | 19930308 |
| JP 1993-515334 | A3 | 19930308 |
| WO 1993-EP517 | A | 19930308 |
| US 1993-146028 | A3 | 19931122 |
| US 1996-723425 | A3 | 19960930 |
| US 2000-576824 | A3 | 20000523 |

TI Process for the determination of peptides corresponding to immunologically important epitopes and their use in a process for determination of antibodies, or biotinylated peptides corresponding to immunologically important epitopes, a process for preparing them and compositions containing them

SO PCT Int. Appl., 133 pp.
CODEN: PIXXD2

IN De Leys, Robert

AB Peptides corresponding to immunol. important epitopes (of bacterial or viral proteins) are determined by (1) preparing peptides corresponding to fragments of the protein of interest, (2) biotinylating the peptides, (3) binding the biotinylated peptides to a solid phase via interation with avidin or streptavidin, and (4) measuring antibodies which bind to the individual peptides. Processes for **biotinylation** of the peptides and for determination of antibodies to hepatitis C virus (HCV), to **HIV**, and to HTLV-I and -II are also disclosed. HCV, **HIV**, HTLV-I, and HTLV-II peptide sequences are included. Use of the biotinylated peptides in the process of the invention makes the anchorage of the peptides to a solid support such that it leaves their essential amino acids free to be recognized by antibodies. In studies determining binding of unbiotinylated peptides directly onto the wells of a polystyrene microtiter plate and binding of biotinylated peptides to wells coated with streptavidin, results demonstrated that antibody binding to the biotinylated peptide is superior to antibody binding to peptide coated directly onto the plastic

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DOCUMENT NUMBER: PREV200100186731
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AUTHOR(S): Li, Min; Yang, Chinglai; Compans, Richard W. [Reprint author]
CORPORATE SOURCE: Department of Microbiology and Immunology, Emory University School of Medicine, Atlanta, GA, 30322, USA
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LANGUAGE: English
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TI Mutations in the cytoplasmic tail of murine leukemia virus envelope protein suppress fusion inhibition by R peptide.

SO Journal of Virology, (March, 2001) Vol. 75, No. 5, pp. 2337-2344. print.
CODEN: JOVIAM. ISSN: 0022-538X.

AU Li, Min; Yang, Chinglai; Compans, Richard W. [Reprint author]

AB During viral maturation, the cytoplasmic tail of the murine leukemia virus (MuLV) envelope (Env) protein undergoes proteolytic cleavage by the viral protease to release the 16-amino-acid R peptide, and this cleavage event activates the Env protein's fusion activity. We introduced Gly and/or Ser residues at different positions upstream of the R peptide in the cytoplasmic tail of the Friend MuLV Env protein and investigated their effects on fusion activity. Expression in HeLa T4 cells of a mutant Env protein with a single Gly insertion after I619, five amino acids upstream from the R peptide, induced syncytium formation with overlaid XC cells. Env proteins containing single or double Gly-Ser insertions after F614, 10 amino acids upstream from the R peptide, induced syncytium formation, and mutant proteins with multiple Gly insertions induced various levels of syncytium formation between HeLa T4 and XC cells. Immunoprecipitation and surface **biotinylation** assays showed that most of the mutants had surface expression levels comparable to those of the wild-type or R peptide-truncated Env proteins. Fluorescence dye redistribution assays also showed no hemifusion in the Env proteins which did not induce fusion. Our results indicate that insertion mutations in the cytoplasmic tail of the MuLV Env protein can suppress the inhibitory effect of the R peptide on membrane fusion and that there are differences in the effects of insertions in two regions in the cytoplasmic tail upstream of the R peptide.

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